

AD-A148 464

HEMATOLOGIC AND BIOCHEMICAL DATA ON HEALTHY INDIVIDUALS
PARTICIPATING IN A. (U) BOSTON UNIV MA SCHOOL OF
MEDICINE E N SERRALLACH ET AL. 28 SEP 83 BUSM-83-14

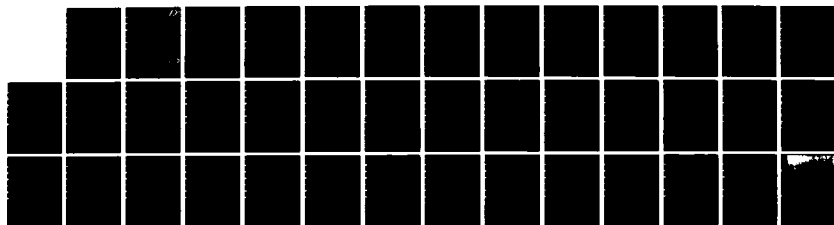
1/1

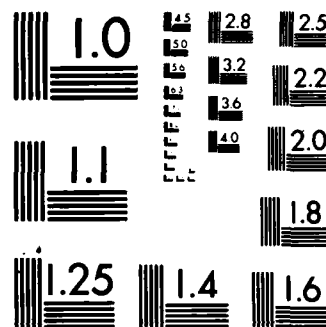
UNCLASSIFIED

N00014-79-C-0168

F/G 6/14

NL





MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS 1963-A

AD A140464

OFFICE OF NAVAL RESEARCH

CONTRACT N00014-79-C-0168

TECHNICAL REPORT NO. 83-14

HEMATOLOGIC AND BIOCHEMICAL DATA ON HEALTHY INDIVIDUALS PARTICIPATING
IN A PHYSICAL CONDITIONING PROGRAM

by

E. N. SERRALLACH,* R. C. DENNIS,* C. RUSHIN,* J. HAY,* M. KLEIN,[†]
W. J. GILLESPIE,^{††} C. P. EMERSON,* AND C. R. VALERI*

*NAVAL BLOOD RESEARCH LABORATORY
BOSTON UNIVERSITY SCHOOL OF MEDICINE
AND

[†]CARDIOVASCULAR DIVISION
UNIVERSITY HOSPITAL
BOSTON UNIVERSITY MEDICAL CENTER
AND

^{††}CARDIOVASCULAR HEALTH AND EXERCISE CENTER
NORTHEASTERN UNIVERSITY
BOSTON, MA 02115

28 SEPTEMBER 1983

DTIC
ELECTE
S D
APR 25 1984
D

Reproduction in whole or in part is permitted for
any purpose of the United States Government.

Distribution of this report is unlimited.

DTIC FILE COPY

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER NBRL, BUSM 83-14	2. GOVT ACCESSION NO. AD A140464	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) HEMATOLOGIC AND BIOCHEMICAL DATA ON HEALTHY INDIVIDUALS PARTICIPATING IN A PHYSICAL CONDITIONING PROGRAM		5. TYPE OF REPORT & PERIOD COVERED Technical Report
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Eugene N. Serrallach, Richard C. Dennis, Carol Rushin, James Hay, *Michael Klein, †W. Jay Gillespie, Charles P. Emerson, C. Robert Valeri		8. CONTRACT OR GRANT NUMBER(s) N00014-79-C-0168
9. PERFORMING ORGANIZATION NAME AND ADDRESS Naval Blood Research Laboratory Boston University School of Medicine 615 Albany St., Boston, MA 02118		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS Naval Medical Research and Development Command Bethesda, Maryland 20814		12. REPORT DATE 28 September 1983
		13. NUMBER OF PAGES 36
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office) Naval Medical Command Department of the Navy Washington, D. C. 20372		15. SECURITY CLASS. (of this report) Unclassified
		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) Approved for public release and sale. Distribution unlimited.		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES *Cardiovascular Division, University Hospital, Boston University Med. Ctr. †Cardiovascular Health and Exercise Center, Northeastern University, Boston, MA 02115		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) Physical fitness Sports anemia Blood Cholesterol Conditioning programs Weight loss Cardiovascular conditioning		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Fifty-eight (58) healthy relatively sedentary adults who were recruited into a physical fitness program involving 10 weeks of closely supervised, standardized and monitored exercises, were evaluated, physically, hemato- logically, and biochemically before and after participation in the program. Most of the participants were subjected to 16 different laboratory tests both before and after participation; a smaller group was subjected to as many as 20 tests. Physical responses to the conditioning program included a loss of body fat averaging 5%, a 3% loss of total body weight, a 32%		

DD FORM 1473

JAN 73

EDITION OF 1 NOV 68 IS OBSOLETE
S/N 0102-LF-014-6601

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

excess body weight loss, decreased pulse rates at peak exertion of 5% and following the recovery period of 11%, an increased aerobic capacity (V02 max) of 8%, and an increased rate of sit-up performance of 28%.

Statistically significant changes in laboratory data included drops in total hemoglobin and MCHC, increased red cell P50 and LDH, decreased serum cholesterol and triglycerides, increased serum HDL-cholesterol, and decreased plasma phosphorus.

The possible significance of these findings is discussed.

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

ABSTRACT

Fifty-eight (58) healthy relatively sedentary adults who were recruited into a physical fitness program involving 10 weeks of closely supervised, standardized and monitored exercises, were evaluated, physically, hematologically, and biochemically before and after participation in the program. Most of the participants were subjected to 16 different laboratory tests both before and after participation; a smaller group was subjected to as many as 20 tests. Physical responses to the conditioning program included a loss of body fat averaging 5%, a 3% loss of total body weight, a 32% excess body weight loss, decreased pulse rates at peak exertion of 5% and following the recovery period of 11%, an increased aerobic capacity (V_{O_2} max) of 8%, and an increased rate of sit-up performance of 28%. Statistically significant changes in laboratory data included drops in total hemoglobin and MCHC, increased red cell P50 and LDH, decreased serum cholesterol and triglycerides, increased serum HDL-cholesterol, and decreased plasma phosphorus.

The possible significance of these findings is discussed.

Accession For	
NTIS GRA&I	<input checked="" type="checkbox"/>
DTIC TAB	<input type="checkbox"/>
Unannounced	<input type="checkbox"/>
Justification	
By	
Distribution/	
Availability Codes	
Dist	Avail and/or Special
A1	



INTRODUCTION

Physical fitness appears to protect individuals against the development of cardiovascular disease,¹⁻³ and therefore is universally considered a desirable goal. Exploring the possibility that improved physical fitness and, by inference, lessened vulnerability to cardiovascular disease might be reflected in the morphology or biochemical composition of the peripheral blood, we have collected and analyzed blood samples from 58 healthy adults before and after a supervised ten-week cardiovascular conditioning program. Tests have included blood cell counts, measurements of mean red cell volume, hemoglobin concentration and red blood cell electrophoretic mobility; red cell 2,3 diphosphoglycerate, adenosine triphosphate, glutamate oxaloacetic transaminase and lactic dehydrogenase levels; serum cholesterol, triglyceride and high density lipoprotein-cholesterol; and plasma lactate, creatine, calcium and phosphorus concentrations.

METHODS

Conditioning Program

The subjects were healthy volunteers aged 21 to 58 (mean age 40), including 44 males and 14 females, who were enrolled in a professionally prescribed aerobic exercise program at the Northeastern University Cardiovascular Health and Exercise Center.⁴ Prior to and following his or her participation in the program each volunteer underwent a complete examination, cardiopulmonary evaluation, graded exercise treadmill test⁵ with a serial EKG evaluation and an aerobic capacity determination, pulmonary function tests, body composition assessment, and a battery of hematologic and biochemical tests (see Table II). Subjects were expected to maintain their individual diets and were enjoined from smoking for the duration of the program. Supervised one-hour classes were conducted three times a week for ten weeks. Each session involved a 15-minute warm-up, which included static stretching and muscular endurance exercises, followed by jogging at a prescribed intensity for 20-40 minutes and ending with a ten-minute cool-down exercise. Each participant's exercise program was individually prescribed at 70-85% of the difference between maximal and resting heart rates, based on the initial graded exercise treadmill test.

Physical Measurements

Calculations of ideal and excess body weight were based on estimates of percent body fat, derived from caliper measurements of skin-fold thickness over the biceps and triceps muscles and in the subscapular and suprapubic

areas, as well as on the individual's height, weight, age and sex.⁵ The pulse rate, determined electrocardiographically prior to, during and following a rigidly standardized graded exercise treadmill test (GXT), based on a modified Bruce protocol,⁶ was recorded for purposes of comparison at "Stage 4" (walking speed, 3.4 MPH; uphill grade, 14%) and after a six-minute walking (1.7 MPH) recovery period at the conclusion of this exercise. The maximum oxygen uptake ($\dot{V}O_2$ max), reflecting peak energy requirements on the GXT, was calculated for each individual in accordance with the following formula: $\dot{V}O_2$ (ml/kg/min.) = (M/min. X % grade X 1.8 X 0.5) + (M/min. X 0.2 + 3.5).⁵ Another criterion of physical fitness (although not of cardiovascular fitness) employed in this study was the maximum number of sit-ups per minute performed prior to and following his or her participation in the conditioning program.

Blood Sampling and Processing

Venous blood samples, collected from all subjects during their initial and final medical examination, were anticoagulated with heparin or CPD, and transported to the Naval Blood Research Laboratory for immediate testing and for cryopreservation to permit simultaneous analysis, at a later date, of the pre-post conditioning samples from each subject.

Freeze-Preservation of Red Cells For Delayed Analysis - Small Aliquot Freezing^{7,8}

Each 10 ml sample of venous blood anticoagulated with 1.4 ml of CPD was stored for up to two hours at room temperature, then packed to a hematocrit reading of about 90%. After removal of the buffy coat the packed cells were well mixed, and a 3 ml sample was transferred to a Corning 50 ml centrifuge

tube with a screw cap. Six ml of a 6.2 M glycerol solution* containing 0.03 g KCl/dl and 1.6 g/dl sodium lactate and buffered with Na_2HPO_4 to a pH of 7.0 were then added in two steps. First, a 1.2 ml aliquot sample of this solution was added dropwise with constant mixing; finally, ten minutes later, the remainder was slowly added to the red cells, again with constant mixing. After 10 minutes of equilibration the screw cap was reapplied, and the tube placed in a -80°C mechanical freezer for storage.

Deglycerolization of Red Blood Cells

The pre- and post-conditioning samples were thawed simultaneously by placement in a 37°C circulating water bath for exactly ten minutes. Red cell washing was done directly in the glycerolization storage tube. Slowly, and with constant mixing, a one-quarter volume of saline solution (2.25 ml of 12% NaCl, pH 6.7-7.4) was added to one volume (9 ml) of a solution containing 0.9% NaCl, 0.2% glucose and 12.5 molar sodium phosphate at pH 6.7, and the mixture again allowed to equilibrate for 2 minutes. The sample was then centrifuged for 2 minutes at 2200 rpm, the supernatant was decanted and the cells were resuspended, using a Vortex magnetic stirrer. The red cells were washed three additional times, each time with 9 ml of the NaCl-glucose-phosphate solution.

Biochemical Analyses

Venous blood gases were measured by conventional electrodes (Instrumentation Laboratory, Model 813), and percent saturation and carbon monoxide were measured in a CO-Oximeter (Instrumentation Laboratory, Model 282).

*Cytosol Laboratories, Boston, MA

Hemoglobin levels were measured with a Coulter hemoglobinometer and hematocrit readings obtained by the microhematocrit centrifugal method. Red cell 2,3 DPG and ATP concentrations were assayed fluorometrically,^{9,10} and plasma lactate was measured spectrophotometrically.¹¹ Red cell P₅₀, defined as the oxygen tension at which hemoglobin is 50% saturated, was measured by the Hemoscan (Aminco) method,¹² and corrected to a pH of 7.2. Small freeze-preserved cell samples with three levels of P₅₀ were used to quality control this measurement.⁸

Red cell glutamic oxaloacetic transaminase (GOT) and lactate dehydrogenase (LDH) activities were measured in lysates which were prepared as follows: Freeze-preserved red cells, deglycerolized and washed as described above, were resuspended to a hematocrit value of about 40%. Red cell counts were obtained with a Model ZF Coulter Counter (aperture 100 microns, amplification 1, threshold 6, aperture current 1) recording computer-adjusted values obtained with a Coulter Model MHR. Red cell lysates equivalent to 100,000 red cells/ μ l were made by adding the appropriate volume of washed red cells to distilled water. The hemoglobin concentration in each lysate was measured by the cyanmethemoglobin method. GOT and LDH levels were obtained by means of a micro-centrifugal analyzer (Instrumentation Laboratory) and reported in international units per microgram hemoglobin.^{13,14}

Plasma creatine was measured spectrophotometrically by a method described originally by Tanzer and Gilvarg;¹⁵ plasma calcium as described by Tietz,¹⁶ using Gilford Diagnostics chromogenic calcium reagent; and plasma inorganic phosphorus by the method of Daly and Ertingshausen¹⁷ employing Gilford Diagnostics inorganic phosphorus reagent.

Measurements of serum cholesterol, triglycerides and high density lipoprotein-cholesterol (HDL) were performed with the Technicon SMAC instrument;* cholesterol by the colorimetric method of Liebermann and Burchard;¹⁸ triglycerides by the enzymatic method of Bucolo and David;¹⁹ and HDL-cholesterol by the selective precipitation method of Lopes-Virella et al.²⁰

Red Cell Sizing by Resistive Pulse Spectroscopy

The mean corpuscular volume of red cells collected from 16 volunteers prior to and following their conditioning programs was measured with a Coulter H4 particle sizer. All aliquot samples were processed similarly to the extent that all were glycerolized as described above. All pre-conditioning samples were cryopreserved pending completion of the conditioning program in each case, at which time they, together with the corresponding post-conditioning samples likewise frozen from the same individual, were simultaneously thawed, deglycerolized and tested. In addition, one of the pre- and one of the post-conditioning samples were fixed in glutaraldehyde (final concentration 1%) prior to their analysis. The Coulter H4 settings were used as follows: Matching Switch, 20 K; Gain Trim, 8.5; Aperture Size, 50/60 microns; Exclusion Threshold, 6.5; High Resolution Circuitry, "ON"; Counts per Channel, 1000; Maximum Counts per Interval, 512000; 1/Amplification, 2; and 1/Aperture Current, 1.00.

Graphic and Tabular Presentation of Data

The physical data obtained in each of our 58 subjects before starting

*These measurements were carried out by Bioran Medical Laboratories, Cambridge, MA

and after completion of this exercise program are presented graphically in Figs. 1-6: Fig. 1, body fat (percent of body weight); Fig. 2, body weight (pounds); Fig. 3, pulse rate at Stage IV of the GXT; Fig. 4, pulse rate after a six-minute walking recovery period following the GXT; Fig. 5, peak oxygen uptake ($\text{VO}_2 \text{ max}$; ml/kg/min.), reflecting energy expenditure during the GXT; and Fig. 6, the number of sit-ups performed per minute with maximum effort.

Each of these figures is comprised of two main panels. In the upper panel are indicated the absolute values obtained before and after conditioning, depicted in each case by an open circle (pre-) connected by a straight line to a solid dot (post-conditioning value). The post/pre numerical ratio of these values is represented by a single circle immediately below in the lower panel. The identity of each subject is indicated by the protocol number which was assigned sequentially upon his or her inclusion in this study and which has determined the positions of the corresponding circles and dots relative to the abscissa. The same format has been applied in Figs. 7-12, charting certain biochemical parameters, i.e., the P_{50} , red cell 2,3 DPG, ATP, serum cholesterol, triglycerides and high density lipoprotein-cholesterol values, respectively, for all subjects tested. The mean \pm S.D. of all post-conditioning/pre-conditioning ratios combined, and the statistical significance of pre- and post-conditioning differences, based on paired T-tests applied to all subjects in each of these groups, are indicated in the corresponding legends.

Seeking a correlation between the degree of physical fitness achieved and the changes in laboratory findings that occurred in response to the

conditioning program, we identified the eight individuals who appeared to show the most improvement and the eight showing the least. These two groups were distinguished on the basis of their loss of body weight and body fat; on the reduction in their pulse rates at Stage IV and after the six-minute walking recovery phase of the GXT; on the increase in their maximal oxygen uptake at Stage IV; and on the increased speed of sit-ups attained. The most conditioned group comprised eight males, ages ranging from 30 to 58 (mean \pm S.D. = 41 ± 10 years). This group included subjects No. 7, 13, 23, 36, 42, 55, and 56. The eight least conditioned subjects, subjects No. 6, 8, 12, 29, 30, 45, 49, and 51, whose ages ranged from 21 to 54 (mean \pm S.D. = 33 ± 12 years), included three females aged 21, 25 and 26 (subjects No. 29, 30, 45, respectively) (see Figs. 13-15).

The physical data obtained in these 16 individuals before and after conditioning are plotted graphically in Fig. 13 (excess body weight, top, and pulse rates, Stage IV, bottom); in Fig. 14 (pulse rates after six-minute recovery walk phase, top, and VO_2 max at Stage IV of the GXT, bottom); and in Fig. 15, indicating sit-up performance. Each line in these figures, connecting a pre-conditioning value (open circle) with a post-conditioning value (solid dot) is numbered in accordance with the individual's subject No. (see also Figs. 2-6). Except for their mean total body weight the two groups were not distinguishable from each other, or from the group as a whole, on the basis of the physical data obtained prior to conditioning, as shown in Table I. The difference in degree of conditioning between these groups, and the effectiveness of the exercise program on the entire group of 58 subjects, is clear from Table II, which records the mean post-conditioning/

pre-conditioning ratio of body fat, total body weight, excess body weight, the pulse rates at Stage IV and six minutes following the GXT, the $\dot{V}O_2$ max, and the sit-up performance of each.

Table III indicates the effect of the program on 16 hematologic and biochemical variables, including total hemoglobin, red cell hematocrit, mean corpuscular hemoglobin concentration, mean corpuscular volume, P_{50} , 2,3 diphosphoglycerate, adenosine triphosphate, lactate dehydrogenase and glutamic oxaloacetic transaminase; serum cholesterol, triglycerides, high density lipoprotein-cholesterol; and plasma lactate, creatine, calcium and phosphorus. Shown are the mean post/pre conditioning ratios of these data for the most and the least conditioned subjects, and for all subjects tested.

RESULTS

Comparison of the physical data obtained on the 58 subjects participating in this study before and after this conditioning program showed clear-cut evidence of improved physical fitness as indicated by a loss of body fat averaging 5%, total body weight 3% and excess body weight 32%; an average decrease of 5% in the pulse rate at Stage IV and 11% following the walking recovery phase of the GXT; a mean rise in $\dot{V}O_2$ max of 8% and an increased rate of sit-up performance averaging 28%. These data are depicted graphically in Figs. 1-6 and are summarized in Table II.

Of the 16 hematologic and biochemical tests applied to these individuals and recorded in Table III, those showing statistically significant pre-/post-conditioning differences for the group as a whole included the total hemoglobin concentration, which fell an average of 2% ($p < 0.002$), P_{50} which increased 2% ($p < 0.05$), LDH which increased 3% ($p < 0.05$), triglycerides which decreased 13% ($p < 0.002$), high density lipoprotein-cholesterol which rose 13% ($p < 0.001$), and the plasma phosphorus concentration which fell an average of 8% (see Figs. 7-12 and Table III).

The group of eight subjects who were selected as "most conditioned" and the group of eight who apparently were "least conditioned" (see Figs. 13-15) were statistically analyzed and compared to each other and to the group as a whole, seeking a possible correlation between changes in physical status and in laboratory findings. The physical data obtained in these two groups prior to conditioning were virtually indistinguishable, as shown in Table I. These data, obtained following completion of the exercise

program and expressed in terms of post-conditioning/pre-conditioning ratios, are shown in Table II. Their laboratory data, in the form of post-/pre-conditioning ratios, are presented in Table III.

It was noted that the best conditioned group of individuals showed the greatest hemoglobin reductions, and the least conditioned group showed the smallest reductions; this was true of the mean corpuscular hemoglobin concentrations also. The red cell LDH was significantly increased ($p < 0.05$) in the entire group of 17 subjects so tested. The P50 was significantly increased in the most ($n = 5$) but not in the least conditioned group ($n = 5$) and in the group as a whole ($n = 41$).

Carboxyhemoglobin concentrations were measured in 40 subjects before and after conditioning. The percentages were within the normal range ($\text{COHb}/\text{total Hb} \times 100 = 2.0\%$) on both occasions in 33 of these subjects; in the remaining 7, presumably as a result of tobacco smoking, these values ranged from 2.1% to 10.9%. Of interest is the fact that two of the subjects with elevated carboxyhemoglobin levels, i.e., No. 8 and No. 12, with pre-conditioning COHb values of 3.3% and 10.8%, and post-conditioning values of 3.7% and 10.9%, respectively, were among the 8 subjects deemed "least conditioned" (see Figs. 13-15).

Of the lipoprotein analyses only the HDL-cholesterol results, showing substantial elevations in the majority of subjects tested ($n = 57$), highest in the most conditioned and least in the least conditioned group, suggest a correlation between the physical data and these particular laboratory data.

Additional tests were performed before and after conditioning on a number of these subjects, the results of which were within normal limits

and were statistically unaffected by the exercise program. These included platelet counts and Coulter H4 platelet sizing ($n = 19$); red cell viscosity at Hct 80, shear 1 and shear 100 ($n = 35$); red cell electrophoresis ($n = 17$), and measurements of whole blood pH, pCO_2 and percent methemoglobin ($n = 21$).

DISCUSSION

The changes we have observed in red cell parameters concomitant with the improved physical status of our subjects, i.e., decreased whole blood hemoglobin and mean corpuscular hemoglobin concentration, although admittedly not large, are consistent with the findings of others and with the appellation "sports anemia"²¹ in its mildest form. The explanation usually offered to explain the phenomenon is intravascular hemolysis with compensatory increase in the output of young, outsized, incompletely hemoglobinized red cells from the marrow, together with possible diversion of red cell hemin to hypertrophying skeletal muscle. The rise in red cell P50 could be regarded as consistent with this hypothesis.²² The latter, to be sure, receives no support from the results of the red cell GOT assay, which shows no statistically significant increase reflecting the presence of an enlarged reticulocyte population. Nor was macrocytosis (i.e., an elevated MCV) discovered on red cell sizing, as one would anticipate in the presence of reticulocytosis. It should be pointed out, however, that the Coulter H4 technic of sizing tends to underestimate the MCV of hypochromic, i.e., relatively flaccid and deformable red cells as they are drawn through the instrument's 50/60 micron aperture. The validity of the MCHC, in contrast to that of the MCV, is relatively unimpeachable, calculated as it is from two of the most reliable measurements in hematology: the photo-electric hemoglobin determination and the spun hematocrit.

The apparent rise in red cell LDH, which would be more consistent with an aging rather than a youthful red cell population, must be questioned on

the basis of sample size, as in the case of GOT.

The serum lipoprotein pattern changed in response to this exercise program precisely as predicted from numerous reports in the literature,^{23,24} for reasons that remain obscure and with cardiovascular benefits yet to be explained.

The 8% drop in inorganic plasma phosphate observed in the group of 35 subjects so tested is also a matter of conjecture. It was unrelated to plasma pH (initially 7.324 ± 0.055 , post-conditioned, 7.309 ± 0.34 , paired $T > 0.1$, $n = 21$), or to pCO_2 (initially 49.1 ± 6.2 , post-conditioned, 54.1 ± 5.0 , paired $T > 0.05$, $n = 21$). It is possible that at the time of sampling this proportion of plasma phosphorus had diffused into the circulating red cells, contributing to their complement of organic phosphates and accounting for their increased P50.

TABLE 1

PHYSICAL DATA OBTAINED PRIOR TO AND AFTER CONDITIONING IN GROUP OF EIGHT SUBJECTS FINALLY SELECTED ON BASIS OF STATISTICAL CHANGES AS THE MOST CONDITIONED, AND THE EIGHT LEAST CONDITIONED, OF ALL FIFTY-EIGHT SUBJECTS.

Initial and Final Values (Mean + S.D.)

	MOST CONDITIONED; N=8		LEAST CONDITIONED; N=8		ALL SUBJECTS; N=58	
	Initial	Final	Initial	Final	Initial	Final
Body Fat, %	27.5 + 5.2	24.8 + 5.3	25.4 + 4.1	25.4 + 3.9	28.2 + 5.4	26.6 + 5.3
Excess Body Weight, %	10.8 + 4.1	5.1 + 2.7	9.6 + 9.3	9.0 + 8.9	11.8 + 7.4	9.0 + 7.5
Pulse Rate, Stage IV of GXT, BPM	168 + 11	149 + 7	176 + 17	174 + 15	171 + 14	160 + 10
Pulse Rate, 6 Min. Recovery, BPM	126 + 16	108 + 7	129 + 14	119 + 16	125 + 14	112 + 14
V _{O2} Max, mls/Kg/Min	38.9 + 6.2	45.6 + 3.9	42.0 + 6.2	44.0 + 4.8	40.0 + 5.1	43.0 + 4.9
Situps, No./Min.	19.4 + 6.8	27.8 + 8.3	21.9 + 13.5	22.2 + 10.8	22.2 + 7.1	26.8 + 6.2

TABLE II

PHYSICAL DATA DISTINGUISHING THE MOST AND THE LEAST CONDITIONED SUBJECTS[†]
 Post-Conditioning/Pre-Conditioning Ratio, Mean[†] S.D. (Statistical Significance)

	Most Conditioned: N=8	Least Conditioned: N=8	All Subjects, N=58
Body Fat, %	0.90 \pm .07*	1.00 \pm .05(NS)	0.95 \pm .06**
Excess Body Weight, %	0.45 \pm .04*	0.90 \pm .46(NS)	0.68 \pm .41**
Pulse Rate Stage IV of GXT, BPM	0.89 \pm .05**	0.99 \pm .04(NS)	0.95 \pm .08**
Pulse Rate, 6 Min. Recovery, BPM	0.86 \pm .09*	0.93 \pm .09(NS)	0.89 \pm .06**
VO ₂ Max., mls/Kg/Min.	1.21 \pm .15*	1.03 \pm .07(NS)	1.08 \pm .11**
Situps, No./Min.	1.47 \pm .23**	0.91 \pm .20(NS)	1.28 \pm .38**

†

Significance of pre- and post-conditioning difference based on
 paired T-test applied to all subjects in each group.

NS Difference not statistically significant ($p > .05$)

* $p < .01$

** $p < .002$

EFFECT OF CONDITIONING PROGRAM ON LABORATORY
FINDINGS IN THE MOST AND LEAST CONDITIONED SUBJECTS
AND IN ALL SUBJECTS TESTED

Test Post-/Pre-Conditioning Ratios: Mean \pm S.D. (Statistical Significance)[†]

	Most Conditioned	Least Conditioned	All Subjects
Hgb	0.35 \pm .04 (p<.02; N=8)*	0.98 \pm .04 (NS; N=8)	0.98 \pm .05 (p<.002; N=58)
Hct	0.99 \pm .04 (NS; N=8)	1.01 \pm .03 (NS; N=8)	1.00 \pm .04 (NS; N=58)
MCHC	0.96 \pm .04 (p<.05; N=8)*	0.99 \pm .04 (NS; N=8)	0.98 \pm .04 (NS; N=57)
MCV	1.00 \pm 0 (NS; N=2)	1.00 \pm .001 (NS; N=3)	1.00 \pm .002 (NS; N=16)
P50	1.02 \pm .02 (p<.05; N=5)*	1.00 \pm .06 (NS; N=5)	1.02 \pm .05 (p<.05; N=41)
2-3 DPG	1.17 \pm .17 (NS; N=5)	1.12 \pm .27 (NS; N=5)	1.03 \pm .21 (NS; N=38)
ATP	0.97 \pm .12 (NS; N=5)	0.99 \pm .23 (NS; N=4)	0.98 \pm .25 (NS; N=38)
LDH	1.02 \pm .04 (NS; N=3)	1.06 \pm .02 (p<.02; N=3)	1.03 \pm .04 (p<.05; N=17)
GOT	1.15 \pm .26 (NS; N=3)	1.05 \pm .06 (NS; N=3)	1.08 \pm .21 (NS; N=17)
Cholesterol	0.92 \pm .10 (NS; N=8)	1.02 \pm .09 (NS; N=8)	0.96 \pm .10 (p<.002; N=58)
Triglycerides	0.80 \pm .33 (NS; N=8)	0.88 \pm .22 (NS; N=8)	0.87 \pm .31 (p<.002; N=58)
HDL	1.25 \pm .24 (p<.02; N=8)*	1.03 \pm .13 (NS; N=8)	1.13 \pm .17 (p<.001; N=57)
Lactate	0.91 \pm .33 (NS; N=5)	0.77 \pm .27 (NS; N=4)	1.03 \pm .31 (NS; N=37)
Creatine	1.14 \pm .26 (NS; N=5)	1.05 \pm .09 (NS; N=4)	1.02 \pm .13 (NS; N=35)
Calcium	0.98 \pm .12 (NS; N=5)	1.05 \pm .09 (NS; N=4)	1.04 \pm .11 (NS; N=35)
Phosphorus	1.02 \pm .24 (NS; N=5)	1.04 \pm .09 (NS; N=4)	0.92 \pm .18 (p<.01; N=35)

†: Significance of pre- and post- conditioning differences based on paired T-test applied to all subjects in each group.

NS: Difference not statistically significant (p>.05).

N: No. of subjects tested.

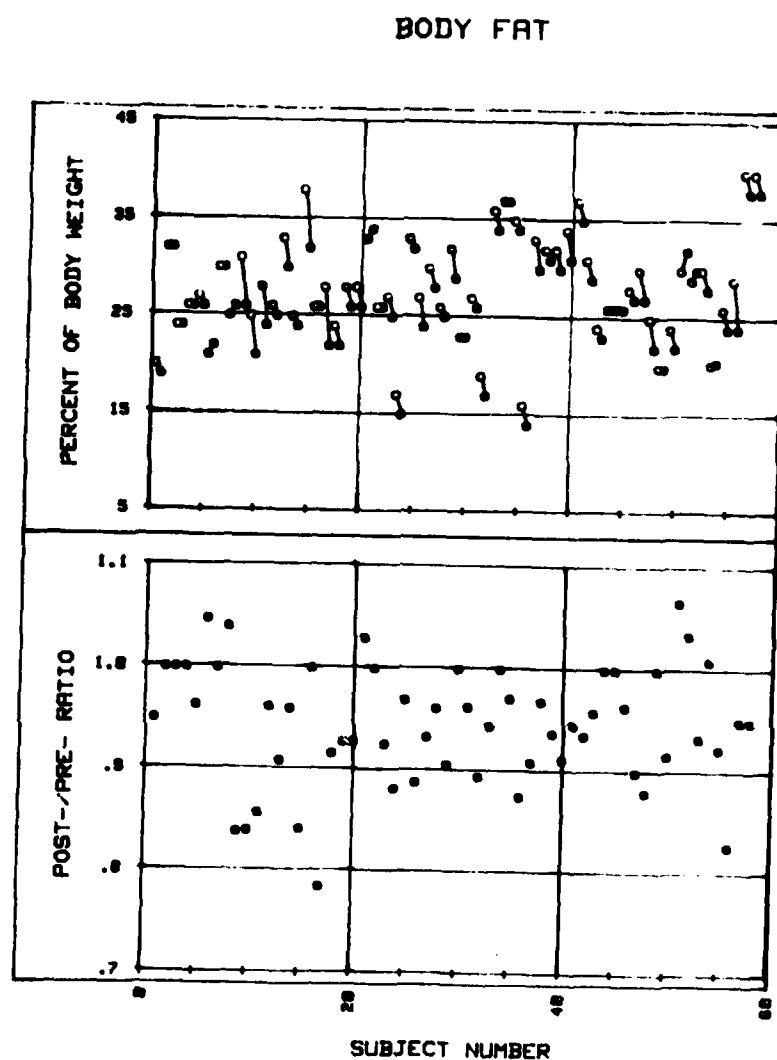


Fig. 1. The mean percentage of body fat prior to conditioning ($M \pm S.D.$) was 28.2 ± 5.4 and the mean post/pre-conditioning ratio, $0.945 \pm .059$. The paired T-test indicated that this fat loss was statistically significant ($p < .002$; $N=58$).

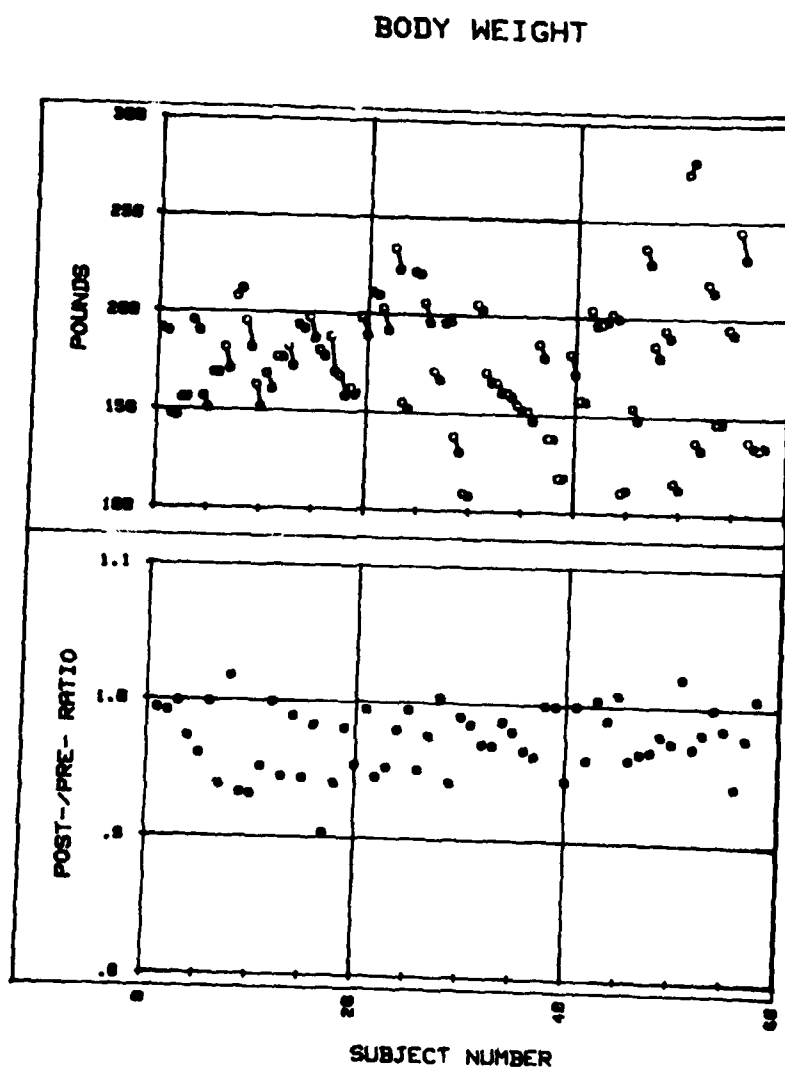


Fig. 2. The mean body weight prior to conditioning ($M \pm S.D.$) was 178.7 ± 33.7 pounds and the mean post/pre-conditioning weight ratio, $0.972 \pm .053$. The paired T-test indicated that this weight loss was statistically significant ($p < .002$; $N=58$).

HEART RATE AT STAGE IV

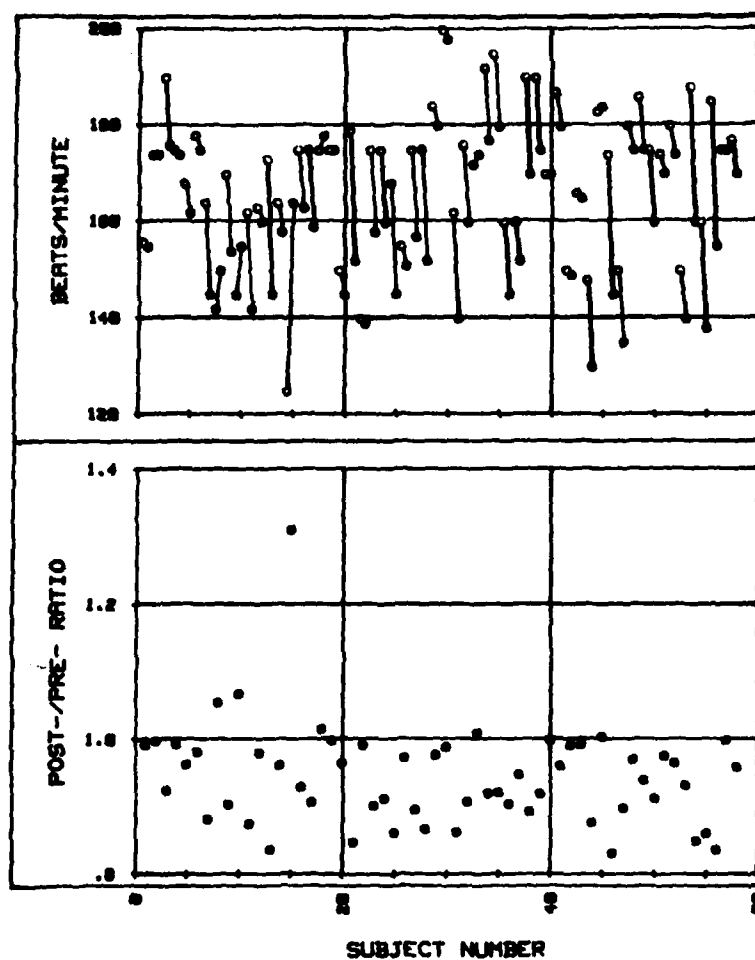


Fig. 3. The mean pulse rate at Stage IV of the GXT prior to conditioning ($M \pm S.D.$) was 171 ± 14 and the mean post/pre-conditioning ratio, $0.946 \pm .075$. The difference was statistically significant ($p < .002$; $N=58$).

HEART RATE, 6 MIN. POST EXERCISE

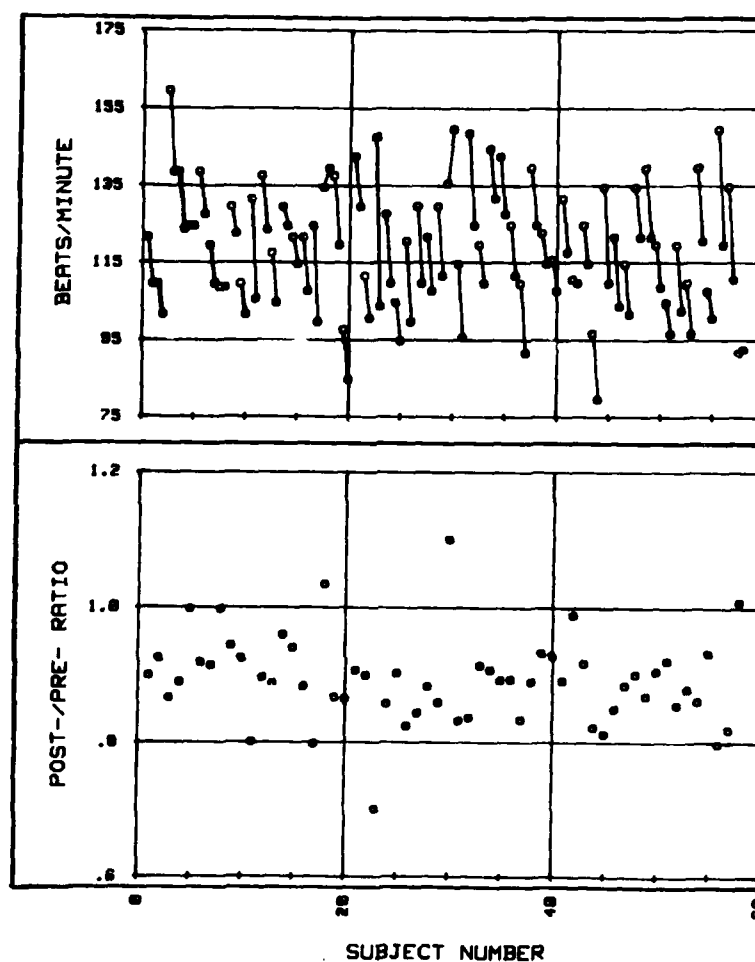


Fig. 6. The mean pulse rate at the end of the 6-minute walking recovery period of the GXT, prior to conditioning, was 125 ± 14 and the mean post/pre-conditioning ratio was $0.895 \pm .064$. This difference was statistically significant ($p < .001$; $N=58$).

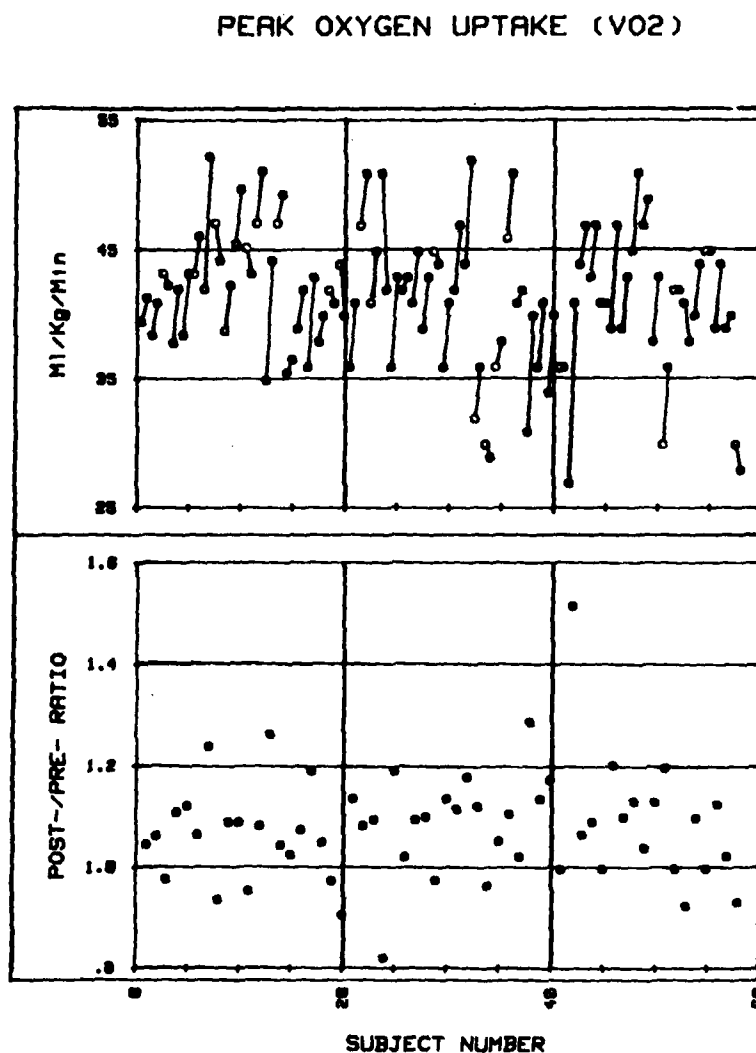


Fig. 5. The mean peak oxygen uptake (VO_2 Max) during the GXT, prior to conditioning, was $40.0 \pm 5.1 \text{ ml/Kg/min.}$ and the post/pre-conditioning ratio, $1.083 \pm .108$. This increase was statistically significant ($p < .001$; $N=58$).

SIT-UPS

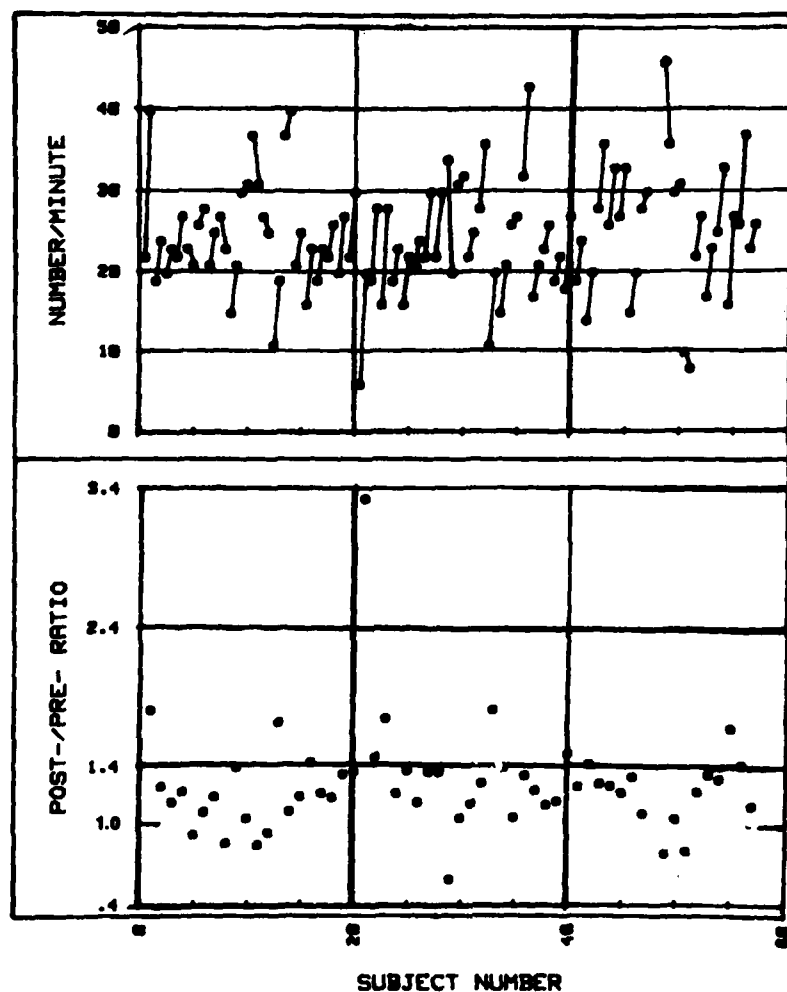


Fig. 6. The mean number of sit-ups performed per minute before conditioning was 20.2 ± 7.1 and the post/pre-conditioning ratio, $1.278 \pm .375$. This increase was statistically significant ($p < .001$; $N=58$).

P50

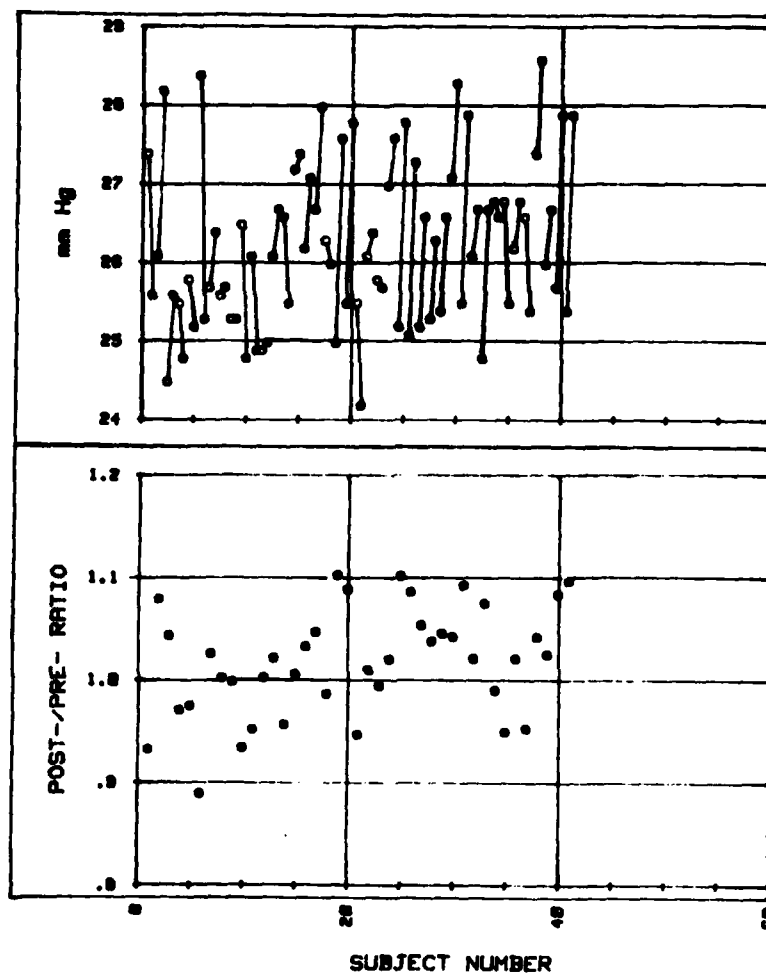


Fig. 7. The mean P50 value before conditioning was 26.0 ± 0.83 and the mean post/pre-conditioning ratio, $1.020 \pm .053$. This increase was statistically significant ($p < .05$; $N=41$).

RED CELL DPG

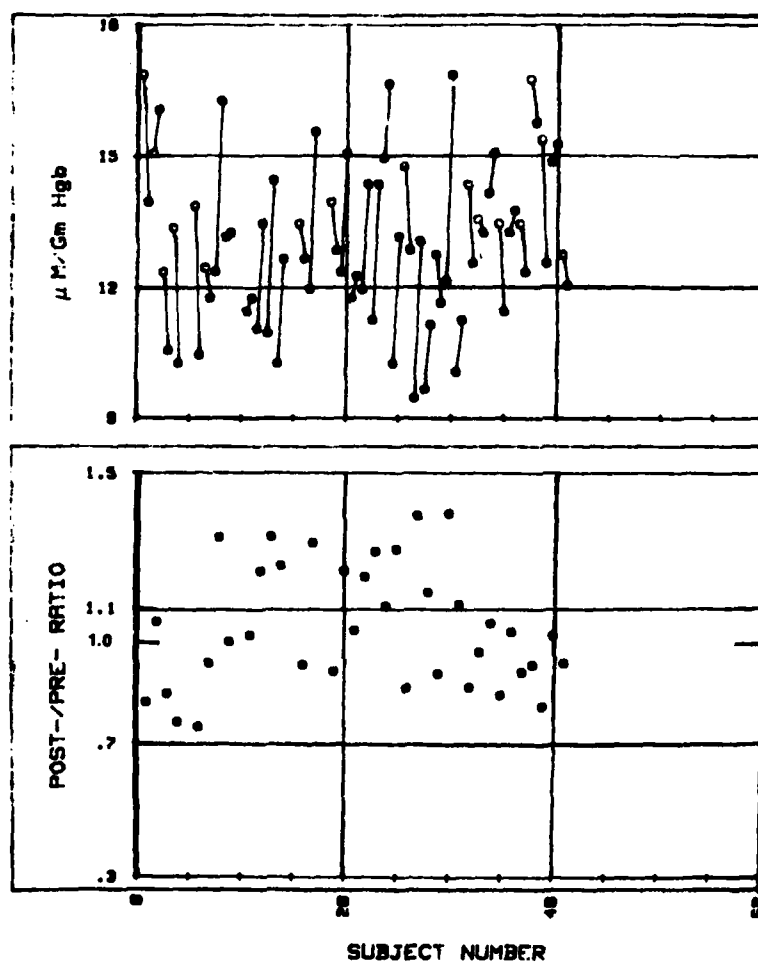


Fig. 8. The mean DPG concentration before conditioning was $12.9 \mu\text{M/Gm Hgb}$ and the mean post/pre-conditioning ratio, 1.035 ± 0.206 . The difference was statistically not significant ($p > 0.20$; $N=37$).

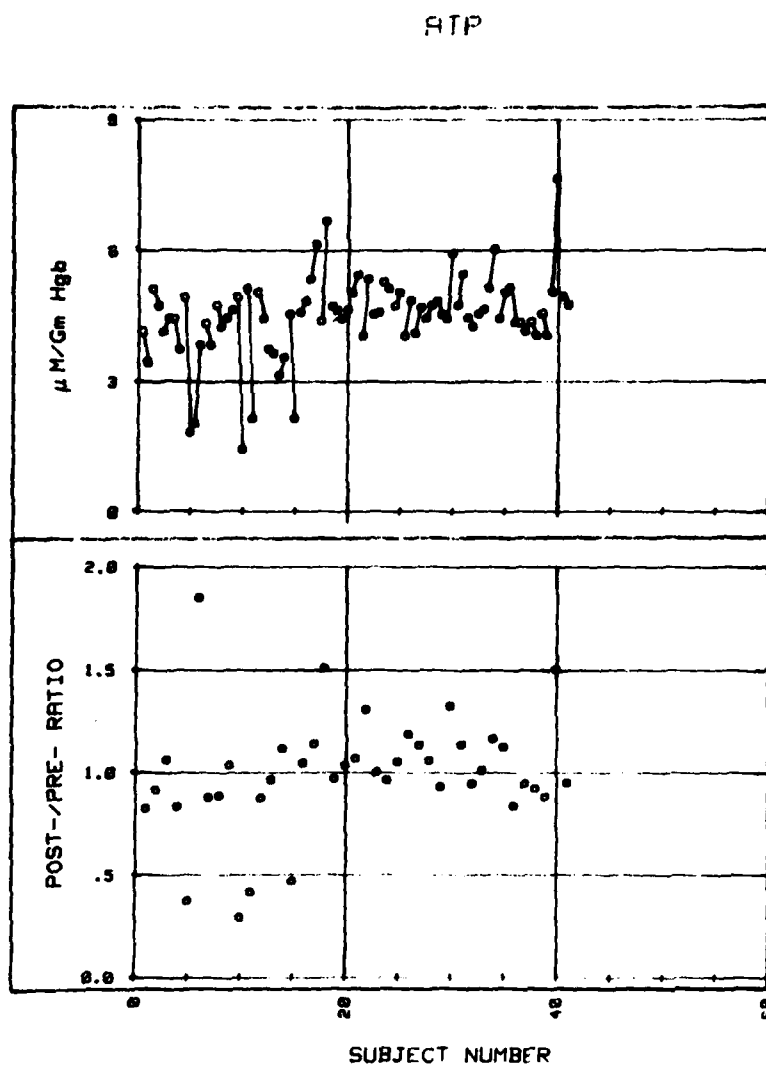


Fig. 9. The mean red cell ATP concentration before conditioning was 4.63 ± 0.45 $\mu\text{M/Gm Hgb}$ and the post/pre-conditioning ratio, 1.007 ± 0.287 . The difference was statistically not significant ($p > 0.5$; $N=38$).

SERUM CHOLESTEROL

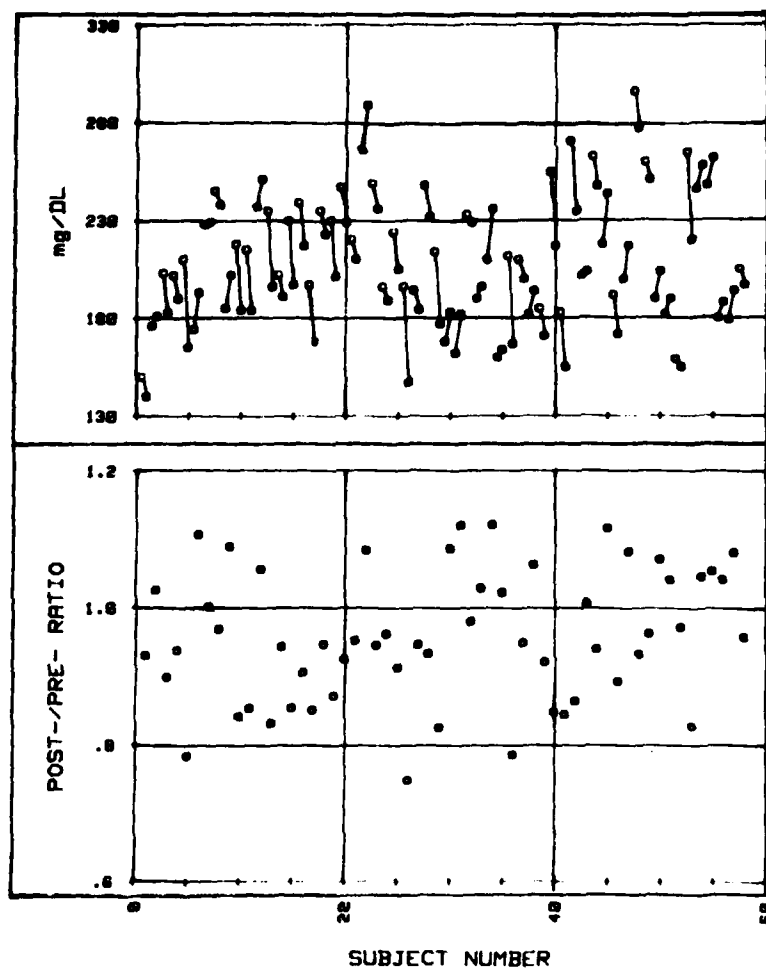


Fig. 10. The mean serum cholesterol concentration before conditioning was 214 ± 30 mg/DL and the mean post/pre-conditioning ratio, 0.961 ± 0.096 . This decrease was statistically significant ($p < .002$; $N=58$).

TRIGLYCERIDES

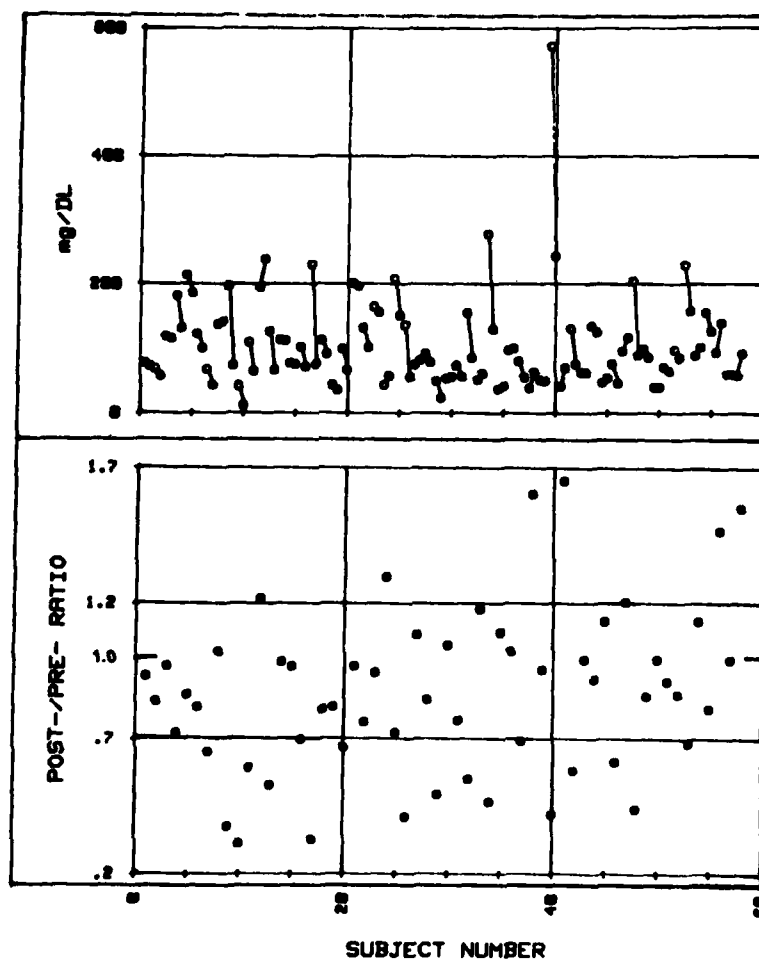


Fig. 11. The mean serum triglyceride concentration before conditioning was 120 ± 85 mg/DL and the mean post/pre-conditioning ratio, 0.873 ± 0.307 . This decrease was statistically significant ($p < .002$; $N=58$).

HIGH DENSITY LIPOPROTEINS

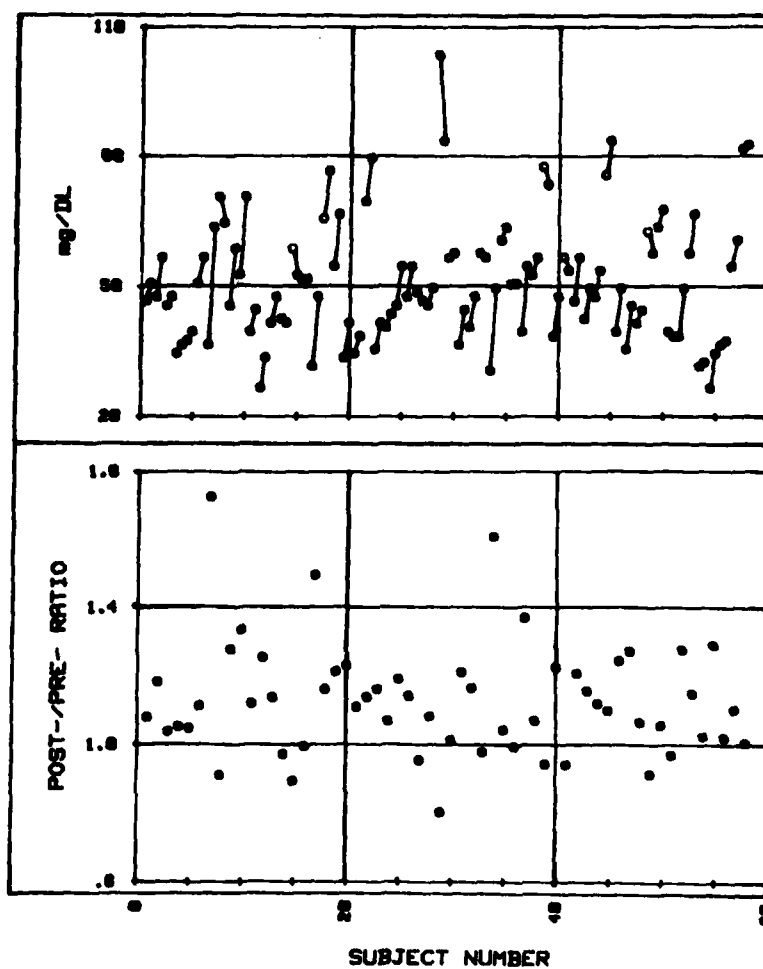


Fig. 12. The mean HDL concentration before conditioning was 49.9 ± 14.8 mg/DL and the mean post/pre conditioning ratio, 1.133 ± 0.165 . The increase was statistically significant ($p < .001$; $N=57$).

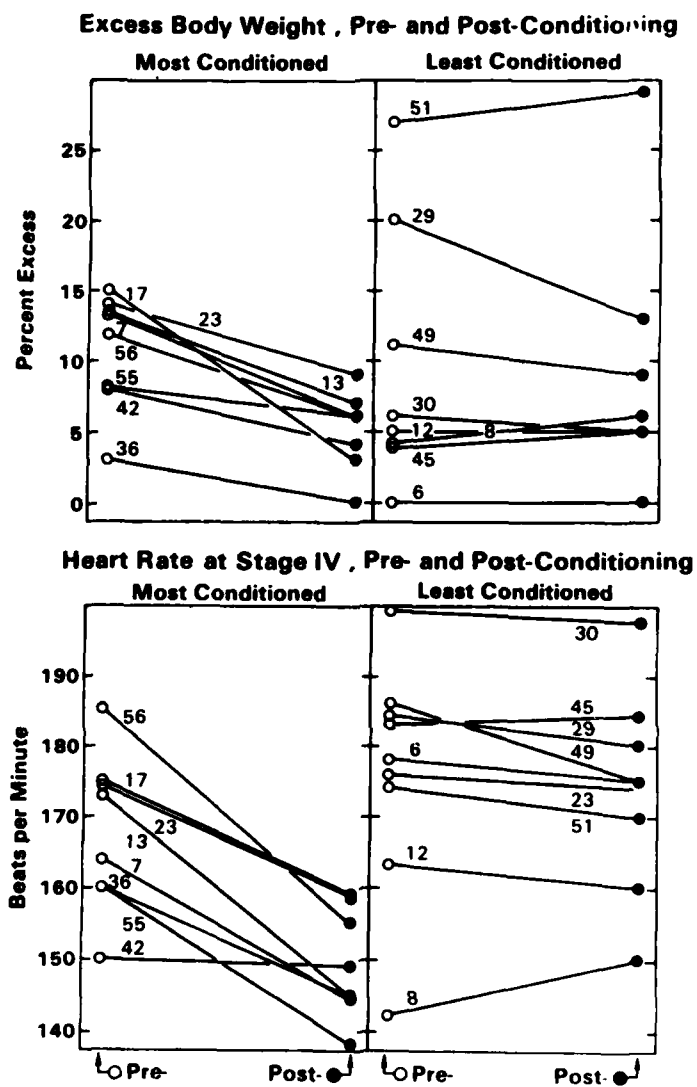


Fig. 13. See text, Figs. 2 and 3, and Tables I and II.

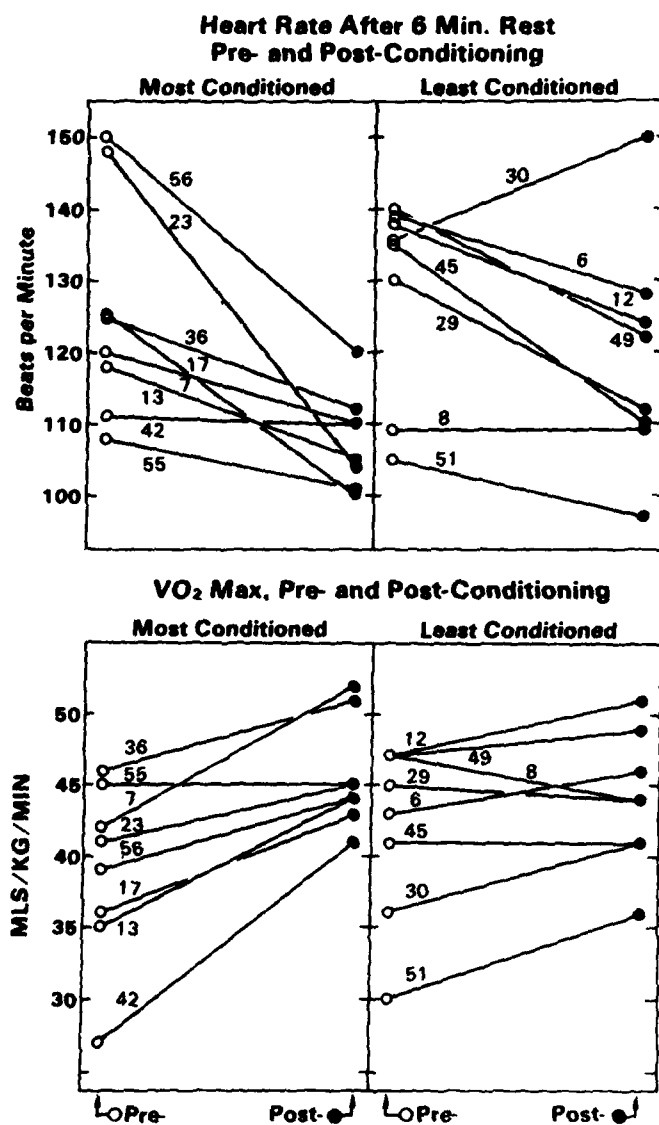


Fig. 14. See text, Figs. 4 and 5, and Tables I and II.

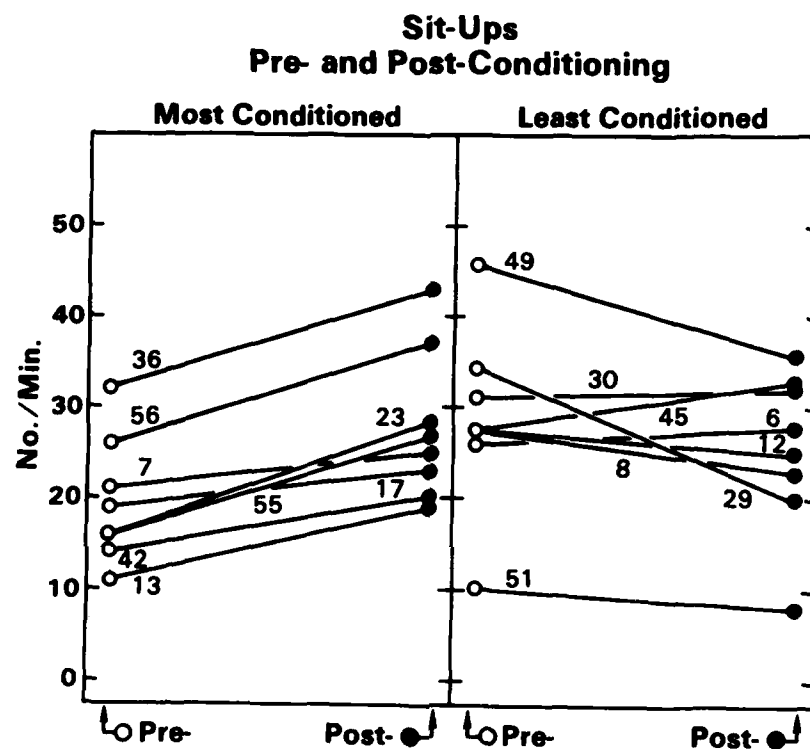


Fig. 15. See text, Fig. 6 and Tables I and II.

REFERENCES

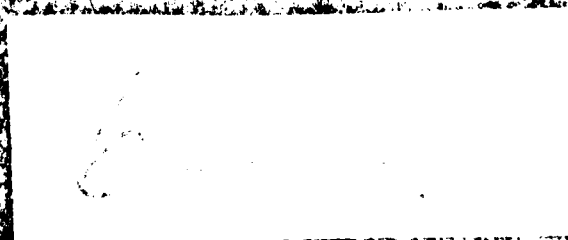
1. Paffenbarger, R. S., Jr., and W. E. Hale: Work activity and coronary heart mortality. *New Engl. J. Med.* 292:545-550, 1975.
2. Morris, J. N., S. P. W. Chave, C. Adam, C. Sirey, L. Epstein, and D. J. Sheehan: Vigorous exercise in leisure time and the incidence of coronay heart disease. *Lancet* 1:333-339, 1973.
3. Williams, R. S., E. E. Logue, J. L. Lewis, T. Barton, N. W. Stead, A. G. Wallace, and S. V. Pizzo: Physical conditioning augments the fibrinolytic response to venous occlusion in healthy adults. *New Engl. J. Med.* 302:987-991, 1980.
4. Northeastern University Brochure: Cardiovascular health and exercise center. Northeastern University Publishing Group, 1980.
5. American College of Sports Medicine: Guidelines for Graded Exercise Testing and Exercise Prescription, 2nd ed., Lea and Febiger, Philadelphia, 1980. Chap. 2 and Appendix F.
6. Bruce, R. A., F. Kusumi, and D. Hosmer: Maximal oxygen intake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am. Heart J.* 85:546-562, 1973.
7. Valeri, C. R. and C. G. Zaroulis: Rejuvenation and freezing of outdated stored human red cells. *New Engl. J. Med.* 287:1307-1313, 1972.
8. Valeri, C. R., D. A. Valeri, R. C. Dennis, J. J. Vecchione, and C. P. Emerson: Biochemical modification and freeze-preservation of red blood cells: A new method. *Crit. Care Med.* 7:439-447, 1979.
9. Keitt, A.: Pyruvate kinase deficiency and related disorders of red cell glycolysis. *Am. J. Med.* 41:762-785, 1966.
10. Lamprecht, W. and I. Trautschold, in Hans-Ulrich Bergmeyer: *Method of Enzymatic Analysis*. Academic Press, N.Y., 1965, 2nd printing, rev. pp. 543-558.

11. Beutler, E.: Red Cell Metabolism: A Manual of Biochemical Methods. Grune and Stratton, N.Y., 1971, pp. 109-111.
12. Dennis, R. C., D. Bechthold, and C. R. Valeri: In vitro measurement of P₅₀ - the pH correction, and use of frozen red blood cells as controls. Crit. Care Med. 7:385-390, 1979.
13. Amador, E., M. F. Massod, and R. J. Franey: Reliability of glutamic-oxalacetic transaminase methods. Am. J. Clin. Path. 47:419-428, 1967.
14. Gay, R. J., R. B. McComb, and G. N. Bowers, Jr.: Optimum reaction conditions for human lactate dehydrogenase isoenzymes as they affect total lactate dehydrogenase activity. Clin. Chem. 14:740-753, 1968.
15. Tanzer, M. L. and C. Gilvarg: Creatine and creatine kinase measurement. J. Biol. Chem. 234:3201-3204, 1959.
16. Tietz, N. W. Fundamentals of Clinical Chemistry. Saunders, Philadelphia, 1970, p. 911.
17. Daly, J. A. and G. Ertingshausen: Direct method for determining inorganic phosphorus in serum with the "Centrifichem". Clin. Chem. 18:263-265, 1972.
18. Levine, J., S. Morgenstern, and D. Vlastelica: A direct Liebermann-Burchard method for serum cholesterol. Automation in Analytical Chemistry, Technicon Symposia, 1967, White Plains, N.Y., Mediad Inc. (1968), pp. 25-28.
19. Bucolo, G. and H. David: Quantitative determination of serum triglycerides by use of enzymes. Clin. Chem. 19(5):475-482, 1973.
20. Lopes-Virella, M. F., P. Stone, S. Ellis, and J. A. Colwell: Cholesterol determination in high-density lipoproteins separated by three different methods. Clin. Chem. 23:882-884, 1977.
21. Radonski, M. W., B. H. Sabiston, and P. Isoard: Development of "sports anemia" in physically fit men after daily sustained submaximal exercise. Aviat. Space Environ. Med. 51:41-45, 1980.

22. Braumann, K. M., D. Boning, and F. Trost: Oxygen dissociation curves in trained and untrained subjects. *Eur. J. Appl. Physiol.* 42:51-60, 1979.
23. Masarei, J. A. L., J. E. Pyke, and F. S. Pyke: Physical Fitness and plasma HDL cholesterol concentrations in male business executives. *Atherosclerosis* 42:77-83, 1982.
24. Wood, P. D., W. Haskell, H. Klein, S. Lewis, M. P. Stern, and J. W. Farquhar: The distribution of plasma lipoproteins in middle-aged male runners. *Metabolism* 25:1249-1257, 1976.

END

FILMED



DTIC